Supreme Court, U.S.
FILED
JUN 1 1990
JOSEPH F. SPANIOL, JR.
CLERK

No. 89-1215

IN THE

Supreme Court of the United States

OCTOBER TERM, 1989

INTERNATIONAL UNION, UNITED AUTOMOBILE, AEROSPACE AND AGRICULTURAL IMPLEMENT WORKERS OF AMERICA, *et al.*,
Petitioners,

v.

JOHNSON CONTROLS, INC.,
Respondent.

On Writ of Certiorari to the United States Court of Appeals for the Seventh Circuit

---

BRIEF OF NATURAL RESOURCES DEFENSE COUNCIL, INC. AS AMICUS CURIAE IN SUPPORT OF PETITIONERS

THOMAS O. MCGARITY
Counsel of Record
University of Texas
School of Law
727 E. 26th Street
Austin, Texas 78705
(512) 471-5151

ALBERT H. MEYERHOFF
90 New Montgomery
San Francisco, CA 94105
Attorneys for Amicus Curiae

WILSON - EPES PRINTING CO., INC. - 789-0096 - WASHINGTON, D.C. 20001

TABLE OF CONTENTS

	Page
INTERESTS OF AMICUS	1
SUMMARY OF ARGUMENT	2
ARGUMENT:	
THE COURT OF APPEALS ERRED IN CONCLUDING THAT SCIENTIFIC DATA FROM EXPERIMENTS WITH LABORATORY ANIMALS ARE TOO SPECULATIVE TO SUPPORT CONCLUSIONS ABOUT THE REPRODUCTIVE TOXICITY OF CHEMICALS IN HUMAN MALES	3
A. An Established Body of Scientific Opinion Supports the Use of Laboratory Animal Studies in Assessing the Reproductive Toxicity of Chemicals	5
B. Courts and Administrative Agencies Have Consistently Relied Upon Laboratory Animal Studies in Assessing the Risks of Chemicals to Humans	12
CONCLUSION	20

TABLE OF AUTHORITIES

CASES: Page

Bell v. Goddard, 366 F.2d 177 (7th Cir. 1966) 16
Environmental Defense Fund, Inc. v. Costle, 578 F.2d 337 (D.C. Cir. 1978) 15
Environmental Defense Fund, Inc. v. EPA, 510 F.2d 1292 (D.C. Cir. 1975) 15
Environmental Defense Fund, Inc. v. EPA, 548 F.2d 998 (D.C. Cir. 1976) 15
Environmental Defense Fund, Inc. v. EPA, 598 F.2d 62 (D.C. Cir. 1978) 15
Environmental Defense Fund, Inc. v. Ruckelshaus, 439 F.2d 584 (D.C. Cir. 1971) 15
Hercules, Inc. v. EPA, 598 F.2d 91 (D.C. Cir. 1978) 15
Industrial Union Dept. AFL-CIO v. American Petroleum Inst., 448 U.S. 607 (1980) 14
International Union v. Johnson Controls, Inc., 886 F.2d 871 (7th Cir. 1989) 4, 7, 16
Society of Plastics Industry v. OSHA, 509 F.2d 1301 (D.C. Cir. 1975) 15
Synthetic Organic Chem. Mfrs. Assn. v. Brennan, 503 F.2d 1155 (3rd Cir. 1974) 15

STATUTES AND REGULATIONS:

16 C.F.R. § 1500.17(a)(10) (1989) 12
29 C.F.R. § 1990.111(a) (1989) 14
29 C.F.R. § 1990.143(g) (1989) 14
40 C.F.R. § 61, Subpart F (1989) 13
40 C.F.R. § 154.7(a)(2)(i) (1989) 12
40 C.F.R. § 261.11(a) (1989) 14
40 C.F.R. § 798 (1989) 13
45 C.F.R. § 46 (1989) 5
38 Fed. Reg. 10929 (1973) 14
39 Fed. Reg. 35890 (1974) 14
40 Fed. Reg. 69532 (1975) 13
41 Fed. Reg. 30476-77 (1976) 13
42 Fed. Reg. 6532 (1977) 13
43 Fed. Reg. 11514 (1978) 16
43 Fed. Reg. 12308-12310 (1978) 12
43 Fed. Reg. 52952 (1978) 17

TABLE OF AUTHORITIES—Continued

Page

44 Fed. Reg. 68624 (1979) 12
50 Fed. Reg. 1122 (1985) 16
50 Fed. Reg. 10372 (1985) 6, 7
53 Fed. Reg. 24834 (1984) 17
53 Fed. Reg. 24850 (1988) 6, 17
54 Fed. Reg. 9386 (1989) 6, 17
7 U.S.C. § 136-137y (1988) 12
15 U.S.C. §§ 1261, 1262 (1988) 12
15 U.S.C. § 2079(a) (1988) 12
15 U.S.C. §§ 2601-2629 (1988) 13
21 U.S.C. § 348(c)(3)(A) (1988) 12
29 U.S.C. § 655 (1988) 14
33 U.S.C. § 1317(a) (1988) 13, 15
42 U.S.C. § 300(f) *et seq.* (1982) 12
42 U.S.C. § 6421 (1982) 14
42 U.S.C. § 6901 *et seq.* (1982) 14
42 U.S.C. § 7412 (1982) 13

OTHER AUTHORITIES:

Equal Employment Opportunity Commission, Policy Guidance on *United Auto Workers v. Johnson Controls, Inc.* 19
Legator Deposition 10, 16
National Academy of Sciences-National Research Council, Biologic Markers in Reproductive Toxicology (1989) 8, 9
National Academy of Sciences-National Research Council, Risk Assessment in the Federal Government: Managing the Process (1983) 6
National Academy of Sciences-National Research Council, Principles for Evaluating Chemicals in the Environment (1975) 6
National Academy of Sciences-National Research Council, Evaluating the Safety of Food Chemicals (1970) 6
I. Nisbet & N. Karch, Chemical Hazards to Human Reproduction (1983) 8
Note, *Exclusionary Employment Practices in Hazardous Industries: Protections or Discriminations?* 5 Colum. J. Env. L. 97 (1978) 10

IN THE

Supreme Court of the United States

OCTOBER TERM, 1989

No. 89-1215

INTERNATIONAL UNION, UNITED AUTOMOBILE, AEROSPACE AND AGRICULTURAL IMPLEMENT WORKERS OF AMERICA, *et al.*,
Petitioners,

v.

JOHNSON CONTROLS, INC.,
Respondent.

On Writ of Certiorari to the United States Court of Appeals for the Seventh Circuit

---

BRIEF OF NATURAL RESOURCES DEFENSE COUNCIL, INC. AS AMICUS CURIAE IN SUPPORT OF PETITIONERS

INTERESTS OF AMICUS[1]

The Natural Resources Defense Council (NRDC), a nonprofit corporation whose principal place of business is at 122 East 42nd Street, New York, N.Y., has offices in Washington, D.C. and San Francisco, California, and a

---

[1] All parties have consented to the submission of this brief. Written letters of consent will be on file with the Clerk.

membership of more than 125,000 persons across the United States and in several other nations. NRDC is dedicated to the perservation, enhancement and defense of the human environment and natural resources of the United States. In carrying out these objectives, NRDC has undertaken efforts to obtain clean air, clean water and toxic substances control, including activities to ensure effective implementation of health and environmental protection statutes. In connection with these activities, NRDC has on numerous occasions relied upon and supported governmental reliance upon laboratory animal studies to assess the risks that exposure to chemicals poses to humans.

SUMMARY OF ARGUMENT

The Court of Appeals erred in concluding that scientific data from experiments with laboratory animals are too speculative to support conclusions about the reproductive toxicity of chemicals in human males. Scientists routinely use laboratory animal testing to assess the risks that chemicals may pose to human beings. The regulatory agencies that Congress has charged with protecting humans from the harmful effects of chemicals in the environment have consistently adhered to the principle that laboratory animal studies are predictive of human health risks, absent clear proof to the contrary. The Court of Appeals was therefore wrong as a matter of scientific fact, as a matter of law, and as a matter of sound public policy in concluding that laboratory animal evidence of the reproductive toxicity of lead in human males was "speculative and unconvincing."

ARGUMENT

THE COURT OF APPEALS ERRED IN CONCLUDING THAT SCIENTIFIC DATA FROM EXPERIMENTS WITH LABORATORY ANIMALS ARE TOO SPECULATIVE TO SUPPORT CONCLUSIONS ABOUT THE REPRODUCTIVE TOXICITY OF CHEMICALS IN HUMAN MALES.

In upholding Respondent Johnson Controls' "business necessity" defense, both the District Court and the Court of Appeals rejected Petitioner International Union's argument that the risks to unborn children posed by worker exposure to lead in the workplace is not limited to females.[2] The International Union offered expert testimony to establish that male worker exposure to lead in the workplace also poses a substantial risk to unborn children. Although the International Union's experts did not attempt to prove conclusively that any particular human malformity had resulted from any particular father's exposure to lead in the workplace, they concluded *inter alia* on the basis of scientific studies conducted with laboratory animals that the exposure of human males to lead in the workplace posed a risk of genetic damage to their offspring. The Court of Appeals found, apparently as a matter of law, that this expert testimony would be inadequate, even if fully accepted by the factfinder, to support a conclusion that male worker exposure to lead

---

[2] The Court of Appeals addressed the question of the usefulness of animal studies in evaluating reproductive risks in the context of the "business necessity" defense, which that court found applicable, despite the fact that the defendant's employment practices clearly constituted disparate treatment. If this Court accepts Petitioners' argument (and the position of Judge Easterbrook below) that in this case of disparate treatment the employer has the burden of proving that the employment practice constitutes a bona fide occupational qualification, then the court need not reach the question of the appropriateness of animal studies for assessing human risks.

posed substantial risks to unborn children. In relevant part, the Court of Appeals reasoned as follows:

> . . . [T]he UAW witnesses posited that animal studies had demonstrated that there was a possible risk of genetic damage to human offspring as a result of male lead exposure. The UAW witnesses attempt to bridge the wide chasm between the results of animal studies and a conclusion of genetic harm allegedly transmitted through the male human being with human studies merely establishing a correlation between male lead exposure and changes in sperm shape. It is interesting to note that the UAW has not presented any medical evidence in the record of any human study scientifically documenting genetic defects in human beings resulting from male lead exposure. It is this lack of convincing scientific data that the plaintiffs attempt to gloss over and cast aside in ignoring the differences between the effect of lead on the human and animal reproductive systems.
>
> . . . Unlike the record evidence of a substantial risk of harm resulting to an unborn child from exposure to lead through the mother's blood stream and placenta, the evidence of risk to the unborn child resulting from exposure of the father to the lead levels currently present in Johnson Controls' battery manufacturing factories is, at best, speculative and unconvincing. The UAW's animal research evidence does not present the type of solid scientific data necessary for a reasonable factfinder to reach a nonspeculative conclusion that a father's exposure to lead presents the same danger to the unborn child as that resulting from a female employee's exposure to lead. . . .

International Union v. Johnson Controls, Inc., 886 F.2d 871, 889-90 (7th Cir. 1989). This statement is erroneous as a matter of law, as a matter of sound public policy, and as a matter of scientific analysis.

A. An Established Body of Scientific Opinion Supports the Use of Laboratory Animal Studies in Assessing the Reproductive Toxicity of Chemicals.

Scientists attempting to assess the toxicity of chemicals to human beings have a limited number of testing options. They can, of course, expose a group of human beings to various predetermined doses of the chemical being tested, observe the effects of the chemical on the exposed humans and their offspring, and compare the responses of the exposed humans to humans in a "control" group of unexposed, but otherwise equivalent humans. Obviously, when a chemical is suspected of producing toxic effects in humans or their offspring, this kind of controlled human testing raises grave moral questions. Past experiences with abuses of human experimentation have led federal agencies to restrict severely the conditions under which federally sponsored human experiments may take place in this country.[3] Even if human testing were ethically allowable, it would take several exposed groups containing thousands of individual humans to draw conclusions about the effects of low exposures (of the sort ordinarily encountered in the workplace) with any degree of statistical confidence. Obviously, such studies would be exceedingly burdensome and expensive.

In the absence of controlled human experiments, the next best source of scientific data on the human toxicity of chemicals is an epidemiological study in which a group of humans who have historically been exposed to greater than normal amounts of the chemical is isolated and compared to an equivalent "control" group. A whole host of methodological problems typically plague epidemiological studies of workplace chemicals. For example, it is often very difficult to determine the historical exposure levels of the workers in the exposed group. Poor medical rec-

[3] See 45 C.F.R. pt. 46 (1989) (Department of Health and Human Services Regulations on the Protection of Human Subjects).

ords also make it difficult to ascertain the incidence of particular diseases and to determine the cause of particular deaths. Selection and information biases can confound attempts to analyze the data objectively. Finally, because most epidemiological studies can examine only a limited number of subjects, only very strong effects can be confidently isolated.[4]

Because of the serious limitations that plague human testing and human epidemiological studies, scientists test chemicals in laboratory animals to discover their toxic effects. Although scientists generally agree that laboratory rodents are not identical in all regards to human beings, there is also general agreement that the results of laboratory animal toxicity testing are relevant to assessing the risks of human exposures to the tested substances. According to the widely-quoted Report of the National Academy of Sciences' Committee on the Institutional Means for Assessment of Risks to Public Health: "The inference that results from animal experiments are applicable to humans is fundamental to toxicological research; this premise underlies much experimental biology and medicine. . . ."[5] The Carcinogen Guidelines of the

[4] See United States Environmental Protection Agency, Proposed Guidelines for Assessing Male Reproductive Risk and Request for Comments, 53 Fed. Reg. 24850, 24861 (1988); United States Environmental Protection Agency, Proposed Amendments to the Guidelines for the Health Assessment of Suspect Developmental Toxicants; Request for Comments; Notice, 54 Fed. Reg. 9386, 9386-99 (1989). *See generally* National Academy of Sciences-National Research Council, Principles for Evaluating Chemicals in the Environment 126 (1975); National Academy of Sciences-National Research Council, Evaluating the Safety of Food Chemicals 44 (1970); Office of Science and Technology Policy, Chemical Carcinogens: A Review of the Science and its Associated Principles, 50 Fed. Reg. 10372, 10379-80 (1985).

[5] National Academy of Sciences-National Research Council, Risk Assessment in the Federal Government: Managing the Process 22 (1983).

Federal Office of Science and Technology Policy, which reflect the combined efforts of senior scientists from all of the major federal regulatory and research agencies, start with the proposition that

> it is reasonable to treat an animal carcinogen as if it were a human carcinogen. . . . This principle has been accepted by all health and regulatory agencies, and is widely regarded by scientists in industry and academia as a justifiable and necessary inference.[6]

These strong statements by panels of experts drawn from the most prestigious members of the scientific community contradict the sugestion in the affidavits of Johnson Controls' paid experts (untested by cross-examination or peer review) that the results of animal studies "were not scientifically established as being applicable to humans." *International Union v. Johnson Controls, Inc.*, 886 F.2d 871, 889 (7th Cir. 1989). The Court of Appeals erred in relying on the affidavits of the experts hired by Johnson Controls for the proposition that the International Union's "animal research evidence does not present the type of solid scientific data necessary for a reasonable factfinder to reach a nonspeculative conclusion. . . ." *Id.* at 889.

In particular, animal studies are relevant (indeed, often critical) to the assessment of the reproductive risks that chemicals in the workplace environment pose to human beings. Nisbet and Karch, for example, devote a chapter of their book "Chemical Hazards to Reproduction" to "Concordance Between Reported Effects in Humans and Measured Effects in Animals." The authors report that "[a] basic assumption in toxicology is that effects observed in experimental animals can be used to infer their likely effects (or lack of effects) in humans, with appropriate consideration of the biological differ-

[6] Office of Science and Technology Policy, *Chemical Carcinogens: A Review of the Science and its Associated Principles*, 50 Fed. Reg. 10372 (1985).

ences between species."[7] After examining the human and animal data for a selected group of chemicals suspected of causing reproductive effects, the authors conclude:

> Teratogenic effects in humans are matched by teratogenic effects in one or more species of experimental animal, and are also matched by the effects of similar risks in at least one species. . . . [T]here may be a fairly close concordance in the dose levels at which humans and animals respond to those agents, humans appearing somewhat more susceptible than the most sensitive animal species tested. In addition, . . . there may be similar concordance between humans and animals for other types of effects on reproduction, although in at least one case an animal species appeared to be much more sensitive than humans.[8]

A National Academy of Sciences expert panel began its discussion of the use of biologic markers in reproductive toxicology with the observation that "[e]xtrapolations to humans are to be based on the most sensitive animal species tested, barring clear evidence that the species is toxicologically distinct from humans."[9]

The Court of Appeals may also have misunderstood the reason that animal studies typically use one or more dose levels that are considerably higher than the level to which humans are exposed in the workplace. Testing animals at high doses is simply a matter of practical necessity. To detect effects that are significant in large exposed populations requires either testing at higher dose levels or testing an impractical number of animals. One National Academy of Sciences panel explained:

---

[7] I. Nisbet & N. Karch, Chemical Hazards to Human Reproduction 93 (1983).

[8] *Id.* at 111.

[9] National Academy of Sciences-National Research Council, Biologic Markers in Reproductive Toxicology 3 (1989).

> The study of reproduction and development poses major resource and logistic problems for those working with laboratory animals. For instance, manageable sample populations do not reveal increases in toxic events of less than 5 to 10%. For some health effects associated with reproduction and neurodevelopment, such as mutagenesis and teratogenesis, incidences in a human population of 3 per 10,000 are significant. Obviously, these effects cannot be well defined in whole-body studies of thousands of experimental animals at a time. Classic toxicology studies of rodents involve the exposure and pathological analyses of 200 animals for 2 years. In such assays, each animal is a surrogate for 1,000,000 people. Birth defects undetectable in this rodent population could be epidemic within ten generations, if they occurred in humans.[10]

In this connection, it is important to note that Dr. Silbergeld (one of the plaintiffs' experts) testified emphatically in her deposition that "[t]here are no clear thresholds for any of these effects of lead." Silbergeld Deposition at 17. This testimony, which must be taken as true for purposes of a motion for summary judgment, indicates that high dose animal studies are relevant for assessing low dose human risks. It is therefore prudent to assume that, in the absence of a demonstrated threshold if lead causes adverse reproductive effects in laboratory animals at high doses, it will pose some risk of adverse effects in humans even at substantially lower exposure levels.

The Court of Appeals apparently failed to recognize that workplace chemicals such as lead can affect fetal development and the health of the subsequently born child in several ways.[11] After a fetus is in existence,

---

[10] National Academy of Science-National Research Council, Biologic Markers in Reproductive Toxicology 4 (1989).

[11] This failure on the part of the Court of Appeals is understandable. Judges have no expertise in toxicology and should there-

it can be harmed by exposure to a chemical substance (transported across the placenta) in much the same way that an adult can be affected, although the fetus may be more susceptible to the chemical's toxic effects than a child or adult. The unborn child can also be damaged prior to conception when a chemical causes mutation in the genetic material of the sperm cell or the egg cell. Although such mutations may not affect the fetus in any significant way, they may well cause adverse effects that car severely affect the life of the human being *post partum*.[12] The first kind of adverse effect only occurs when a pregnant female is exposed to a toxic chemical. The second kind of effect can occur when either a male or a female is exposed. As a general matter, it cannot be said that one effect is more significant than the other, and for that reason it is impossible to conclude that exposure to a chemical that causes only the first kind of effect is riskier than exposure to a chemical that causes only the second kind of effect. Exposure to one chemical may present a high probability of a relatively modest consequence (e.g., slightly reduced I.Q.) while exposure to another chemical may pose a low probability of a disastrous consequence (e.g., hydrocephalus). In the absence of a great deal of information about the relative probabilities and without some very difficult judgments about the relative severity of disease end-points, it is impossible to conclude that one exposure poses a higher risk to the unborn human than the other.

---

fore be cautious about drawing conclusions about the worthiness scientific data. This caution is especially warranted when the scientific information available to the court comes in the form of untested expert testimony in litigation affidavits.

[12] See generally Note, *Exclusionary Employment Practices in Hazardous Industries: Protection or Discrimination?* 5 Colum. J. Env. L. 97, 99-100 (1978). Dr. Legator's desposition testimony, which must be accepted as true for purposes of a motion for summary judgment, was that "lead is one of the very few chemicals . . . that probably causes a genetic lesion during spermatogenesis." Legator Deposition at 20.

The real world is more complex than the above example suggests. Exposure to a single substance may pose a risk of both kinds of adverse effects. Preconception exposure of either the male or female may pose a small probability of mutation leading to a grotesque malformity, while post-conception exposure of the female may lead to more modest health effects. Thus, the International Union's claim with respect to lead exposure in the workplace is not at all fanciful. The post-conception scenario is relatively well-understood. Scientists know that lead passes from the mother's blood to the fetus at some time during pregnancy. Scientists also know that elevated lead levels in children are associated with various diseases ranging in severity from relatively minor adverse effects at low blood levels to more severe effects at high blood levels. The knowledge-base for these relatively strong scientific conclusions consists of human epidemiological studies, a large number of animal studies and occasional clinical observations.

It is much more difficult to explore preconception toxicity with epidemiological studies. Proper epidemiology would require knowledge about both the mother's and father's exposures and about all pregnancy outcomes, even though some mutations result in spontaneous abortions. This information is much harder to acquire. Scientists, regulatory agencies and courts are therefore forced to rely more heavily upon laboratory animal studies as the knowledge-base from which to draw conclusions about the effects of preconception exposure to chemical substances. Reliance on animal studies is concededly more "speculative," but it was wrong for the court to conclude that animal studies are not "convincing" or "solid" scientific data. When existing epidemiological studies do not provide adequate information on human exposures to the substance at issue, it is not at all "unscientific" or "unreasonable" to draw conclusions about human risks from existing animal studies.

B. Courts and Administrative Agencies Have Consistently Relied Upon Laboratory Animal Studies in Assessing the Risks of Chemicals to Humans.

From the beginning of this century, Congress has enacted precatory statutes designed to protect the public health from the toxic effects of chemicals in food, the workplace and the general enivornment. The regulatory agencies that Congress created to implement these statutes have, with the approval and often active encouragement of the reviewing courts, consistently relied upon laboratory animal studies in evaluating human risks. In the last two decades, governmental reliance on laboratory animal studies has become thoroughly interwoven into the regulatory fabric of our society.

A partial list of federal laws in which animal studies are routinely used to assess human health risks includes: the Federal Insecticide, Fungicide and Rodenticide Act;[13] the Federal Food, Drug and Cosmetic Act;[14] the Consumer Product Safety Act;[15] the Safe Drinking Water Act;[16]

---

[13] 7 U.S.C. § 136-137y; EPA routinely relies upon animal data in reaching pesticides regulatory decisions, see, e.g., 40 C.F.R. Part 154.7(a)(2)(i) ("validated test data" used for cancellation decisions, due to carcinogenicity, teratogenicity or reproductive toxicity from "effects demonstrated in humans or experimental animals").

[14] 21 U.S.C. § 348(c)(3)(A) ("no additive shall be deemed to be safe if it is found to induce cancer when ingested by man or animal.")

[15] 15 U.S.C. § 2079(a) and the Federal Hazardous Substances Act § 2(f)(1)(A), 15 U.S.C. § 1261, 1262. See e.g., 16 C.F.R. § 1500.17 (a)(10), and 43 Fed. Reg. 12308-12310 (animal studies are the basis for classification as banned hazardous substance of self-pressurized household substances containing vinyl chloride monomer).

[16] 42 U.S.C. §§ 300(f) *et seq.* See, e.g., 44 Fed. Reg. 68624, National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water:

> . . . sufficient scientific evidence had been accumulated to conclude that chloroform is an animal carcinogen as shown from a properly conducted bioassay and should be presumed to be a risk to humans and that, as such, prudent public health policy warrants reasonable measures to reduce human exposure. . . . In the specific case of chloroform and other THMs, EPA had relied primarily on animal studies demonstrating the toxicology of chloroform. . . . EPA has extrapolated from the results of animal studies to assess the risk posed by THMs to humans.

Id. at 68627.

the Toxic Substances Control Act;[17] the Federal Water Pollution Control Act;[18] the Federal Clean Air Act;[19] the

---

[17] 15 U.S.C. §§ 2601-2629 (1976). § 2603(b)(2)(A) provides:

> The health and evironmental effects for which standards for the development of test data may be prescribed include carcinogenesis, mutagenesis, teratogenesis, behavioral disorders, cumulative or synergistic effects, and any other effect which may present an unreasonable risk of injury to health or the environment. . . . The methodologies that may be prescribed in such standards include epidemiologic studies, serial or hierarchical test, in vitro test, and *whole animal tests*, ...

(Emphasis supplied); see also 40 C.F.R. Part 798 which specifies the use of numerous animal test procedures to determine chemical oncogenicity (§§ 798.3300, 798.320) and reproductive toxicity (§§ 798.5100-798.5955) for purposes of implementing § 4 of the Act.

[18] FWPCA § 307(a), 33 U.S.C. § 1317(a) (1976). See, e.g., 41 Fed. Reg. 30476-77 (1976) and 42 Fed. Reg. 6532 (1977) (proposed and final discarges standards for PCBs based on evidence of animal carcinogenicity).

[19] 42 U.S.C. § 7412. See, e.g. National Emission Standards for Hazardous Air Pollutants: Proposed Standard for Vinyl Chloride, 40 Fed. Reg. 69532 (December 24, 1975). (The rationale for the standard included animal test data demonstrating carcinogenicity of vinyl chloride as well as epidemiological studies.) See also, Nation Emission Standard for Vinyl Chloride, 40 C.F.R. Part 61, Subpart F.

Occupational Safety and Health Act;[20] and the Resource Conservation and Recovery Act.[21]

Under these statutes, the federal courts have uniformly recognized the need to regulate carcinogens and reproductive toxins on the basis of animal test results in the absence of definitive human epidemiological data. The plurality opinion of this Court in the *Benzene* case recognized the practical necessity of basing public policy decisions affecting public health on laboratory animal studies. The opinion observed that several "rational" techniques existed for determining whether human exposure to a toxic substance in the workplace presented a "significant risk" of material health impairment and noted that "[i]n other proceedings, the Agency has had a good deal of data from animal experiments on which it could base a conclusion on the significance of the risk." *Industrial Union Dept., AFL-CIO v. American Petroleum Inst.*, 448 U.S. 607, 657 n.64 (1980). If, as the Court of Appeals in this case maintained, evidence from animal studies is in fact "speculative" and "unconvincing," it would have been disingenuous for this Court to have suggested that

---

[20] 29 U.S.C. § 655. See OSHA Cancer Policy which "establishes the criteria and procedures under which substances will be regulated by OSHA as potential occupational carcinogens." 29 C.F.R. § 1990.111(a). Positive results in high-dosage animal tests "will be used to establish the qualitative inference of carcinogenic hazard to workers." 29 C.F.R. § 1990.143(g). See, e.g., 39 Fed. Reg. 35890, 35891 (1974), 38 Fed. Reg. 10929 (1973) (OSHA exposure standards for vinyl chloride and ethyleneimine, respectively, based on animal studies indicative of carcinogenicity).

[21] 42 U.S.C. § 6901 *et seq.* See 42 U.S.C. § 6421 and implementing regulations at 40 C.F.R. 261.11(a):

> The Administrator shall list a solid waste as a hazardous waste [if] . . . (3) it contains any of the toxic constituents listed in Appendix VIII . . . Substances will be listed in Appendix VIII only if they have been shown in scientific studies to have toxic, carcinogenic, mutagenic or teratogenic effects on humans *or other life forms.*

(Emphasis supplied).

OSHA could meet the Court's "significant risk" test by relying solely upon such evidence.

The United States Court of Appeals for the District of Columbia has observed that the government

> has a 'heavy burden' to 'explain the basis for . . . [a] decision to permit the continued use of a chemical known to produce cancer in experimental animals.' [citations omitted]. When firm evidence establishes that a chemical is a carcinogen, statutes leave an administrator no alternative but to step in and protect the public.'

Environmental Defense Fund, Inc. v. EPA, 598 F.2d 62, 88 (D.C. Cir. 1978). See also *Environmental Defense Fund, Inc. v. EPA*, 510 F.2d 1292 (D.C. Cir. 1975) (upholding the ban of the pesticides aldrin and dieldrin based upon carcinogenicity in rats and mice); *Environmental Defense Fund, Inc. v. Ruckelshaus*, 439 F.2d 584, 590 (D.C. Cir. 1971) (DDT); *EDF v. Costle*, 578 F.2d 337, 345 (D.C. Cir. 1978) (court remanded to EPA regulations implementing the Safe Water Drinking Act that failed adequately to protect the public upon "ample evidence . . . our drinking water is contaminated with a large variety of organic substances, of demonstrated carcinogenicity in animals"); *Environmental Defense Fund, Inc. v. EPA*, 548 F.2d 998, 1005-06 (D.C. Cir. 1976), cert. denied, 431 U.S. 925 (1977) (heptachlor and chlordane) (court approved EPA's use of "cancer principles" that "accept the use of animal test data to evaluate human cancer risks; [and] consider a positive carcinogenic effect in test animals as sufficient to characterize a pesticide as posing a cancer risk to man . . ."); *Hercules Inc. v. EPA*, 598 F.2d 91 (D.C. Cir. 1978) (EPA regulation of toxaphene and endrin under section 307 of the Clean Water Act, 33 U.S.C. § 1317); *Society of Plastics Industry v. OSHA*, 509 F.2d 1301 (D.C. Cir. 1975) (OSHA regulation of vinyl chloride); *Synthetic Organic Chem. Mfrs. Assn. v. Brennan*, 503 F.2d 1155 (3rd Cir. 1974)

(court upheld OSHA's workplace exposure standard for ethyleneimine, upon its demonstrated carcinogenicity in mice and rats, noting "the extrapolation gathered from data in these rodent experiments to humans is justified by the report of the Ad Hoc Committee on the Evaluation of Low Levels of Environmental Chemical Carcinogens to the Surgeon General"); *Bell v. Goddard*, 366 F.2d 177, 180-81 (7th Cir. 1966) (court affirmed an FDA ban on diethelstilbestrol (DES) as a poultry feed supplement on evidence of its carcinogenicity in test animals, noting "there is a histologically remarkable similarity between cancers of the endometrium in humans and animals").

Judge Easterbrook, in his dissenting opinion in the Court of Appeals below, speculated that "[t]he medical profession, like the Food and Drug Administration, will be stunned to discover that animal studies are too 'speculative.'" 886 F.2d at 919. Judge Easterbrook was right. Not only have the medical profession and the Food and Drug Administration consistently relied upon animal studies to assess human health risks, but virtually every other federal agency charged with protecting the public health from hazardous chemicals has also extrapolated from "mouse-to-man" in evaluating human health risks. Indeed, it would be irresponsible for an agency charged with protecting health to do otherwise.[22]

[22] In his deposition testimony Dr. Legator gave an example of an instance in which the Environmental Protection Agency's failure to act on the basis of animal studies on the pesticide DBCP resulted in sterility in workers exposed to that substance:

> With DBCP we had substantial information about the fact that this chemical at five parts per million caused testicular atrophy in a variety of animals. The study was generated in 1963. And basically we ignored the animal data, until the workers found an effect themselves in 1977.

Legator Deposition at 52. Upon discovering the human consequence, EPA cancelled the registration of DBCP. 50 Fed. Reg. 1122 (1985). OSHA promulated a standard to protect workers from DBCP in 1978. 43 Fed. Reg. 11514 (1978).

In promulgating its standard for workplace exposure to lead, the Occupational Safety and Health Administration concluded:

> The record in this rulemaking is clear that male workers may be adversely affected by lead as well as women. Male workers may be rendered infertile or impotent, and both men and women are subject to genetic damage which may affect both the course and outcome of pregnancy. . . .[23]

In forming this conclusion, the agency relied heavily upon the very animal studies that the Court of Appeals in this case summarily rejected on the basis of a summary judgment record devoid of cross-examination, peer review or any other validating technique.

The United States Environmental Protection Agency in 1988 proposed guidelines for assessing male and female reproductive risks[24] and for assessing the risks of suspected developmental toxicants.[25] Both sets of guidelines rely heavily upon data from animal experiments in evaluating human risks. Under the "weight of the evidence" approach that the guidelines adopt for characterizing male reproductive risks due to chemical exposures, the guidelines posit that a "known positive" reproductive effect exists when "[a] convincing body of evidence exists that an agent causes an adverse effect on the male reproductive system in humans." A "probable positive" effect exists when "[a] convincing body of evidence exists that

[23] Occupational Safety and Health Administration, Final Standard for Occupational Exposure to Lead, 43 Fed. Reg. 52952, 52966 (1978).

[24] Environmental Protection Agency, Proposed Guidelines for Assessing Female Reproductive Risk, 53 Fed. Reg. 24834 (1988); Environmental Protection Agency, Proposed Guidelines for Assessing Male Reproductive Risk, 53 Fed. Reg. 24850 (1988).

[25] Environmental Protection Agency, Proposed Amendments to the Guidelines for the Health Assessment of Suspect Developmental Toxicants, 54 Fed. Reg. 9386 (1989).

an agent causes an adverse effect on the male reproductive system in nonhuman mammals." 53 Fed. Reg. at 24863. While it is clear that human testing and epidemiological studies are the strongest evidence of male reproductive effects, the EPA has concluded that animal studies alone are not "specuiative" or "unconvincing" evidence of human reproductive effects.

The Guidelines for Developmental Toxicity address, *inter alia*, the risks that pre- and post-conception exposures pose to unborn humans.[26] One of the fundamental assumptions underlying the guidelines is that

> an agent that produces an adverse developmental effect in experimental animal studies is assumed to pose a potential hazard to humans following exposure during development. This assumption is based on the data for known human developmental toxicants. In almost all cases, the experimental animal data would have predicted a developmental effect in humans.

54 Fed. Reg. at 9390. The guidelines provide that 'adequate evidence of potential human developmental toxicity exists where there is 'data from an appropriate, well-executed study in a single experimental animal species that demonstrates developmental toxicity. . . .'" 54 Fed. Reg. at 9399. It goes without saying that "adequate" evidence is not "speculative" or "unconvincing."

Clearly, the Court of Appeals' conclusion that laboratory animal data do not constitute "convincing scientific data" does not comport with the scientific judgment of

---

[26] The guidelines define "developmental toxicology" as "[t]he study of adverse effects on the developing organism that may result from exposure prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. . . . The major manifestations of developmental toxicity include: "(1) death of the developing organism, (2) structural abnormality, (3) altered growth and (4) functional deficiency." 54 Fed. Reg. at 9390-91.

the agency that Congress has charged with regulating environmental risks to human reproduction.

The Equal Employment Opportunity Commission issued policy guidance to its investigators in the wake of the Court of Appeals' decisions in this case that spoke directly to the use of laboratory animal data to assess human risks in both sexes:

> The majority in *Johnson Controls* rejected UAW's animal research evidence, stating that it was "speculative," "unconvincing" and not "solid scientific data." . . . Commission investigators should not reject animal studies in such a wholesale manner. . . . These studies may in some cases provide the best available evidence to evaluate fetal or other human health hazards in the workplace. Certainly, the data from the animal research in this case, along with the other data provided, should have been sufficient to create a genuine issue of material fact warranting denial of summary judgment.[27]

The Commission was clearly correct in telling its investigators not to fall into the trap that ensnared a majority of Court of Appeals in this case.

The Commission correctly pointed out in its policy guidance that the question of the extent to which human health risk assessment should rely upon animal toxicity data was more properly the domain of the public health agencies like the Environmental Protection Agency and the Occupational Safety and Health Administration.[28]

---

[27] Equal Employment Opportunity Commission, Policy Guidance on *United Auto Workers v. Johnson Controls, Inc.*, reprinted at p. 127a of the Appendix to the Petition for Certiorari.

[28] The Policy Guidance states:

> In determining whether the harm is mediated only through one sex, animal research should be considered along with other objective scientific evidence. The Commission will defer to pertinent findings by government agencies such as OSHA and

The reviewing courts, including this Court, should heed this sage advice. Court are not well qualified, either technically or institutionally, to engage in wholesale science policy judgments of the sort that the Court of Appeals in this case mandated. As we have seen, virtually every federal agency that is charged with implementing public health laws routinely relies upon animal testing data in assessing human health risks. This Court should defer to that expert judgment.

CONCLUSION

The International Union in this case contends that Johnson Controls has the burden of proving that its disparate treatment of women reflects a "bona fide occupational qualification." If this Court accepts this view of the law, then there is no need to address the use of animal studies in evaluating human reproductive risks. To meet the "business necessity" test adopted by the Court of Appeals in this case, however, the International Union offered evidence from laboratory animal studies tending to prove that lead exposure to male humans at levels comparable to those found in the Johnson Controls plant posed a significant risk of adverse effect to unborn children through mutations of the genetic material in sperm cells. The International Union was not allowed to introduce this evidence in its legitimate attempt to persuade the factfinder that the overall risk posed by male exposure is roughly comparable (if not higher) than the risk posed by female exposure through transplacental passage of lead into the blood stream of existing fetuses. In finding that the evidence from animal testing was "speculative" and "unconvincing," the Court of Appeals departed from a long-accepted and well-established practice of scientists,

NIOSH [the National Institute for Occupational Safety and Health].

Appendix to the Petition for Certiorari at 127a.

regulatory agencies and reviewing courts of using animal studies to evaluate human health risks.

Respectfully submitted,

THOMAS O. MCGARITY
Counsel of Record
University of Texas
School of Law
727 E. 26th Street
Austin, Texas 78705
(512) 471-5151

ALBERT H. MEYERHOFF
90 New Montgomery
San Francisco, CA 94105
Attorneys for Amicus Curiae